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Non-obstructive Coronary Arteries After Tenecteplase in STEMI: Clinical and Angiographic Phenotype from a Pharmacoinvasive Network

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Abstract

Background

Non-obstructive coronary arteries may be observed after fibrinolysis for ST-elevation myocardial infarction (STEMI), but their interpretation is challenging because thrombus dissolution may modify subsequent angiographic anatomy. We characterized this post-tenecteplase phenotype within a pharmacoinvasive STEMI network.

Methods

This retrospective cohort included 1,875 patients with STEMI treated with tenecteplase between 2009 and 2016. Non-obstructive coronary arteries (≤ 50% stenosis) were compared with obstructive disease (≥ 70%).

Results

Non-obstructive coronary arteries occurred in 73 patients (3.9%). Compared with obstructive disease, patients were younger (51.6 ± 13.1 vs. 58.1 ± 11.2 years), had lower GRACE scores (121.1 ± 48.0 vs. 143.2 ± 39.0), higher LVEF (55.0 ± 13.0% vs. 48.3 ± 11.4%), greater initial TIMI flow (2.9 ± 0.5 vs. 2.1 ± 1.2), and higher myocardial blush grade (2.7 ± 0.9 vs. 1.6 ± 1.5; all $P < 0.001$). Older age (adjusted OR per 10 years, 0.61; 95% CI, 0.47–0.81), dyslipidemia (OR, 0.50; 95% CI, 0.28–0.91), hs-TnT > 100 pg/mL (OR, 0.22; 95% CI, 0.12–0.39), and LVEF < 50% (OR, 0.45; 95% CI, 0.24–0.83) were independently associated with lower odds of this phenotype.

Conclusions

Non-obstructive coronary arteries after tenecteplase were uncommon and associated with younger age, lower atherosclerotic risk, less myocardial injury, preserved ventricular function, and greater coronary patency. These findings support interpretation as a post-treatment angiographic phenotype rather than a single underlying mechanism.

1. Introduction

Primary percutaneous coronary intervention (pPCI) is the preferred reperfusion strategy for patients with ST-elevation myocardial infarction (STEMI) when it can be performed within guideline-recommended timeframes. However, timely access to pPCI remains challenging in many healthcare systems. In this setting, fibrinolysis remains an established reperfusion alternative when timely pPCI cannot be achieved. [1–3]

The pharmacoinvasive strategy (PIS) combines early fibrinolytic therapy with subsequent transfer to a PCI-capable center. Following successful fibrinolysis, coronary angiography with PCI when indicated is performed within 2–24 h, whereas immediate angiography and rescue PCI are indicated in patients with failed reperfusion.[1–3] Randomized trials, including TRANSFER-AMI and STREAM, have provided important evidence supporting PIS when timely pPCI is not feasible.[4, 5]

Coronary angiography after fibrinolysis allows assessment of coronary anatomy following fibrinolytic therapy. While most patients with STEMI demonstrate significant obstructive coronary artery disease at subsequent angiography, a small proportion have non-obstructive coronary arteries. The clinical and angiographic characteristics associated with this post-fibrinolysis non-obstructive coronary phenotype remain poorly characterized.[6–9]

We therefore investigated patients with STEMI treated with tenecteplase within an organized pharmacoinvasive network who subsequently underwent coronary angiography. We aimed to characterize the clinical and angiographic phenotype associated with non-obstructive coronary arteries (≤ 50% stenosis) after fibrinolysis compared with patients with significant obstructive coronary artery disease, with particular emphasis on markers of epicardial and microvascular reperfusion.

2. Methods

2.1 Study design and population

This retrospective observational cohort study included patients treated within an organized STEMI network in São Paulo, Brazil, between August 2009 and December 2016. Patients presenting with STEMI at referring facilities received fibrinolytic therapy with tenecteplase and were subsequently transferred to a tertiary academic cardiovascular center for invasive coronary angiography as part of a pharmacoinvasive strategy.

According to the network protocol in effect during the study period, coronary angiography was planned 3–24 h after fibrinolysis, with PCI performed when indicated. Patients with evidence of failed reperfusion or other indications for an urgent invasive strategy underwent earlier coronary angiography and rescue PCI when indicated. Clinical information was prospectively collected in the original registry, whereas the present analysis was conducted retrospectively.

Patients aged ≥ 18 years with STEMI treated with tenecteplase were eligible. Patients with non-ST-elevation myocardial infarction, unstable angina, pericarditis, myocarditis, early repolarization with inadvertent fibrinolysis, Takotsubo syndrome, STEMI treated with primary PCI, fibrinolysis with agents other than tenecteplase, or inadequate angiographic data were excluded.

The study was approved by the institutional Research Ethics Committee (approval no. 2.621.563; CAAE 66561717.6.0000.5505). The underlying STEMI registry was registered at ClinicalTrials.gov (NCT02090712).

2.2 Study groups

Patients were classified according to coronary anatomy at angiography following tenecteplase administration. The non-obstructive coronary artery group comprised patients with ≤ 50% stenosis in the presumed culprit coronary artery, whereas the obstructive coronary artery group comprised patients with ≥ 70% stenosis in the presumed culprit vessel.

Patients with intermediate coronary stenosis (51–69%) were not included in the primary comparison between the non-obstructive and obstructive groups. According to the clinical protocol in effect during the study period, these patients were initially managed medically, with subsequent revascularization considered when myocardial ischemia was demonstrated by functional testing, including pharmacological stress myocardial perfusion imaging or stress echocardiography.

2.3 Angiographic assessment

All available coronary angiograms were reassessed using archived digital images. Angiographic interpretation was performed independently by two interventional cardiologists who were unaware of each other's assessment. Patients were included in the non-obstructive group only when agreement regarding stenosis severity was achieved; cases with disagreement were excluded.

The presumed culprit artery was determined by integrating angiographic findings with the electrocardiographic territory and available echocardiographic or ventriculographic findings. Initial and final Thrombolysis in Myocardial Infarction (TIMI) flow grades and initial myocardial blush grade were assessed.[10, 11]

2.4 Clinical variables and outcomes

Demographic characteristics and cardiovascular risk factors included age, sex, diabetes mellitus, hypertension, chronic kidney disease, obesity, dyslipidemia, smoking, family history of cardiovascular disease, previous myocardial infarction, and reported illicit drug use.

Clinical variables included symptom-to-needle time, systolic blood pressure at admission, GRACE, TIMI, and CRUSADE risk scores, high-sensitivity troponin T at admission, and left ventricular ejection fraction assessed by transthoracic echocardiography.

The clinical outcome available for the present analysis was in-hospital all-cause mortality. Long-term outcomes were not analyzed because adequate follow-up was available for only approximately 25% of the original cohort.

2.5 Statistical analysis

Continuous variables were assessed for distribution and reported as mean ± standard deviation or median (interquartile range), as appropriate. Categorical variables were presented as frequencies and percentages. Comparisons between patients with non-obstructive and obstructive coronary arteries

were performed using Student's *t* test or the Mann–Whitney U test for continuous variables and the χ^2 test or Fisher's exact test for categorical variables, as appropriate.

A multivariable logistic regression model was constructed to identify factors independently associated with the presence of non-obstructive coronary arteries after fibrinolysis. Candidate variables were selected based on clinical relevance and univariable association, while model complexity was limited according to the number of patients with non-obstructive coronary arteries. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A two-sided *P* value < 0.05 was considered statistically significant.

3. Results

3.1 Study population

Among 2,161 patients initially evaluated within the STEMI network, 278 were excluded according to the predefined eligibility criteria, leaving 1,883 patients for angiographic assessment. Of these, 81 patients had non-obstructive coronary arteries (≤ 50% stenosis); eight were subsequently excluded because of disagreement regarding angiographic stenosis severity, resulting in 73 patients in the non-obstructive group. The obstructive group comprised 1,802 patients with significant coronary artery stenosis (≥ 70%). The final study population therefore included 1,875 patients, of whom 73 (3.9%) had non-obstructive coronary arteries after fibrinolysis (Fig. 1).

3.2 Clinical characteristics

Patients with non-obstructive coronary arteries were younger than those with obstructive coronary artery disease (51.6 ± 13.1 vs. 58.1 ± 11.2 years; P < 0.001). They had lower prevalences of diabetes mellitus (11.0% vs. 29.9%; P = 0.001), dyslipidemia (35.6% vs. 51.4%; P = 0.012), and previous myocardial infarction (1.4% vs. 9.8%; P = 0.027), whereas male sex (80.8% vs. 69.2%; P = 0.046) and reported illicit drug use (9.6% vs. 3.7%; P = 0.024) were more frequent.

Patients with non-obstructive coronary arteries also had a lower clinical risk profile, with lower GRACE (121.1 ± 48.0 vs. 143.2 ± 39.0; P < 0.001), TIMI (3.2 ± 2.5 vs. 3.9 ± 5.9; P = 0.037), and CRUSADE scores (19.6 ± 13.1 vs. 25.7 ± 14.4; P = 0.001), and higher left ventricular ejection fraction (55.0 ± 13.0% vs. 48.3 ± 11.4%; P < 0.001). No significant between-group differences were observed in hypertension, chronic kidney disease, obesity, smoking, family history of cardiovascular disease, or systolic blood pressure at admission (Tables 1 and 2).

Table 1
Baseline demographic and cardiovascular risk characteristics

Variable	Non-obstructive coronary arteries (n = 73)	Obstructive coronary arteries (n = 1,802)	*P* value
Age, years	51.6 ± 13.1	58.1 ± 11.2	< 0.001
Male sex, n (%)	59 (80.8)	1245 (69.2)	0.046
Diabetes mellitus, n (%)	8 (11.0)	539 (29.9)	0.001
Hypertension, n (%)	42 (57.5)	1099 (61.1)	0.630
Chronic kidney disease, n (%)	5 (6.8)	142 (7.9)	0.918
Obesity, n (%)	14 (19.2)	391 (21.7)	0.711
Dyslipidemia, n (%)	26 (35.6)	924 (51.4)	0.012
Smoking, n (%)	40 (54.8)	1149 (63.9)	0.146
Family history of cardiovascular disease, n (%)	10 (13.7)	435 (24.2)	0.055
Reported illicit drug use, n (%)	7 (9.6)	66 (3.7)	0.024
Previous myocardial infarction, n (%)	1 (1.4)	177 (9.8)	0.027

Data are presented as mean ± standard deviation or n (%), as appropriate. Percentages were calculated using available observations for each variable.

Table 2
Clinical and angiographic characteristics according to coronary anatomy after fibrinolysis

Variable	Non-obstructive coronary arteries (n = 73)	Obstructive coronary arteries (n = 1,802)	*P* value
Symptom-to-needle time, min	386.7 ± 421.4	356.3 ± 616.8	0.012
Systolic blood pressure at admission, mmHg	131.0 ± 32.9	134.1 ± 28.4	0.338
GRACE score	121.1 ± 48.0	143.2 ± 39.0	< 0.001
TIMI risk score	3.2 ± 2.5	3.9 ± 5.9	0.037
CRUSADE score	19.6 ± 13.1	25.7 ± 14.4	0.001
hs-TnT > 100 pg/mL at admission, n (%)	42 (59.2)	1577 (90.2)	< 0.001
LVEF, %	55.0 ± 13.0	48.3 ± 11.4	< 0.001
Initial TIMI flow grade	2.9 ± 0.5	2.1 ± 1.2	< 0.001
Initial myocardial blush grade	2.7 ± 0.9	1.6 ± 1.5	< 0.001

Data are presented as mean ± standard deviation or n (%), as appropriate. Percentages were calculated using available observations for each variable. Abbreviations: CRUSADE, Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes with Early Implementation of the ACC/AHA Guidelines; GRACE, Global Registry of Acute Coronary Events; hs-TnT, high-sensitivity troponin T; LVEF, left ventricular ejection fraction; TIMI, Thrombolysis in Myocardial Infarction.

3.3 Myocardial injury and angiographic characteristics

Markers of myocardial injury and ventricular dysfunction differed substantially between groups. High-sensitivity troponin T > 100 pg/mL at admission was less frequent among patients with non-obstructive coronary arteries (59.2% vs. 90.2%; P < 0.001), whereas left ventricular ejection fraction was higher (55.0 ± 13.0% vs. 48.3 ± 11.4%; P < 0.001).

Patients with non-obstructive coronary arteries demonstrated substantially greater epicardial and microvascular patency at angiography. Mean initial TIMI flow was 2.9 ± 0.5 compared with 2.1 ± 1.2 in obstructive STEMI (P < 0.001), whereas initial myocardial blush grade was 2.7 ± 0.9 vs. 1.6 ± 1.5, respectively (P < 0.001) (Table 2).

3.4 In-hospital mortality

In-hospital mortality occurred in 1 of 73 patients (1.4%) with non-obstructive coronary arteries and in 90 patients (5.0%) with obstructive coronary arteries (P = 0.256) (Fig. 2).

3.5 Multivariable analysis

In multivariable logistic regression analysis, older age was independently associated with lower odds of non-obstructive coronary arteries after fibrinolysis (adjusted OR per 10-year increase, 0.61; 95% CI, 0.47–0.81; P < 0.001). Dyslipidemia was also inversely associated with the non-obstructive phenotype (adjusted OR, 0.50; 95% CI, 0.28–0.91; P = 0.024).

High-sensitivity troponin T > 100 pg/mL at admission (adjusted OR, 0.22; 95% CI, 0.12–0.39; P < 0.001) and LVEF < 50% (adjusted OR, 0.45; 95% CI, 0.24–0.83; P = 0.011) were independently associated with lower odds of non-obstructive coronary arteries. Sex, diabetes mellitus, and reported illicit drug use were not independently associated with the non-obstructive phenotype (Fig. 3 and Supplementary Table S1).

4. Discussion

In this cohort of patients with STEMI treated with tenecteplase within an organized pharmacoinvasive network, non-obstructive coronary arteries at subsequent angiography were uncommon, occurring in 3.9% of the study population. These patients exhibited a distinct clinical and angiographic phenotype characterized by younger age, fewer traditional atherosclerotic risk factors, less myocardial injury, preserved left ventricular systolic function, and greater epicardial and microvascular patency. In multivariable analysis, older age, dyslipidemia, hs-TnT > 100 pg/mL, and LVEF < 50% were independently associated with lower odds of the non-obstructive phenotype, whereas no variable was independently associated with increased odds. These findings suggest that this phenotype may be characterized more by the absence of features associated with established atherosclerotic disease and greater myocardial injury than by a single specific clinical marker.

The interpretation of non-obstructive coronary arteries in this setting requires consideration of the timing of angiography relative to fibrinolytic therapy. Because all patients received tenecteplase before coronary anatomy was assessed, the angiographic findings represent a post-treatment state rather than the coronary anatomy at STEMI presentation. Accordingly, a non-obstructive culprit vessel cannot necessarily be interpreted as evidence that an obstructive lesion or thrombotic occlusion was absent at presentation. Successful fibrinolysis may have restored epicardial patency and reduced thrombus burden before angiography, potentially leaving little residual stenosis. This distinction is particularly relevant when considering these findings within contemporary frameworks for myocardial infarction with non-obstructive coronary arteries.[1, 12]

The markedly greater initial TIMI flow and myocardial blush grade observed in the non-obstructive group support this interpretation. Patients with non-obstructive coronary arteries had a higher mean initial TIMI flow (2.9 vs. 2.1) and myocardial blush grade (2.7 vs. 1.6) than those with obstructive disease. These findings indicate greater epicardial patency and better microvascular reperfusion and are consistent with more effective pharmacological reperfusion. Nevertheless, angiography alone cannot determine whether the underlying mechanism was complete or near-complete thrombus dissolution over a previously disrupted plaque, coronary spasm, coronary embolism, spontaneous coronary artery dissection, microvascular dysfunction, or another cause of STEMI without persistent obstructive disease.[12–15]

The clinical profile of these patients was also consistent with a lower burden of established atherosclerotic disease. Patients with non-obstructive coronary arteries were approximately 6 years younger and had lower prevalences of diabetes, dyslipidemia, and previous myocardial infarction. Importantly, after multivariable adjustment, increasing age and dyslipidemia remained independently associated with lower odds of belonging to the non-obstructive group. Rather than identifying a specific positive predictor of non-obstructive anatomy, these findings suggest that the non-obstructive phenotype becomes less likely as features associated with established atherosclerotic disease become more prominent.[6, 16]

The differences in markers of myocardial injury and ventricular function provide an additional dimension to this phenotype. Patients with non-obstructive coronary arteries were less likely to have hs-TnT > 100 pg/mL and had better preserved LVEF, while both hs-TnT > 100 pg/mL and LVEF < 50% remained independently associated with lower odds of the non-obstructive phenotype. Together with the higher TIMI flow and myocardial blush grade, these findings are consistent with less extensive myocardial injury and better myocardial reperfusion. Whether this reflects earlier or more complete thrombus dissolution, a smaller ischemic burden, different underlying coronary mechanisms, or a combination of these factors cannot be determined from the present data.[11, 17]

Reported illicit drug use was more frequent among patients with non-obstructive coronary arteries in the unadjusted analysis, but this association was no longer observed after multivariable adjustment. Although stimulant drugs such as cocaine may cause myocardial infarction through different coronary mechanisms, including mechanisms without fixed obstructive coronary disease, the unadjusted finding in our cohort may reflect differences in age, cardiovascular risk profile, or other characteristics associated with illicit drug exposure. Given the small number of exposed patients and the lack of information regarding the specific substance, timing, or dose, this observation should be considered hypothesis-generating.[18, 19]

In-hospital mortality was numerically lower among patients with non-obstructive coronary arteries than among those with obstructive disease (1.4% vs. 5.0%), although the difference was not statistically significant. The very small number of deaths in the non-obstructive group precludes firm conclusions regarding prognosis. Nevertheless, this difference is directionally consistent with their younger age, lower clinical risk profile, better preserved LVEF, less myocardial injury, and greater epicardial and microvascular patency. Larger studies with adequate long-term follow-up are required to determine whether this post-fibrinolysis angiographic phenotype is associated with meaningful prognostic differences.

These findings have practical implications for the interpretation of coronary angiography after fibrinolysis. The identification of non-obstructive coronary arteries after tenecteplase should not automatically be considered evidence that fibrinolysis was unnecessary or that the initial STEMI diagnosis was incorrect. In a pharmacoinvasive pathway, the observed coronary anatomy represents a post-treatment state, and successful thrombus dissolution may contribute to the absence of significant

residual stenosis. Conversely, non-obstructive anatomy should not be assumed to represent successful fibrinolysis in every patient, as alternative coronary and non-coronary mechanisms remain possible. When clinically appropriate, further investigation with cardiac magnetic resonance, intracoronary imaging, and functional coronary assessment may help identify the underlying mechanism.[12, 14]

5. Study limitations

Several limitations should be acknowledged. First, this was a retrospective analysis of a prospectively collected registry and is therefore subject to the limitations inherent to observational studies. Second, the non-obstructive group was relatively small, limiting statistical power, particularly for mortality and multivariable analyses. Third, coronary angiography was performed after tenecteplase administration; therefore, pre-fibrinolysis coronary anatomy and thrombus burden were unknown, preventing determination of whether non-obstructive anatomy represented successful thrombus dissolution or another underlying mechanism. Fourth, systematic cardiac magnetic resonance imaging, intracoronary imaging with optical coherence tomography or intravascular ultrasound, and coronary vasoreactivity testing were not available, precluding definitive mechanistic classification of the non-obstructive phenotype. Fifth, LVEF and some laboratory variables were not available for all patients, reducing the effective sample size of the multivariable analysis. Finally, long-term outcomes could not be reliably assessed because adequate follow-up was available for only a minority of the original cohort.

6. Conclusion

In conclusion, non-obstructive coronary arteries after tenecteplase were uncommon among patients with STEMI treated within a pharmacoinvasive network and were associated with a distinct phenotype characterized by younger age, a lower atherosclerotic risk profile, less myocardial injury, preserved ventricular function, and greater epicardial and microvascular patency. Older age, dyslipidemia, elevated hs-TnT, and reduced LVEF were independently associated with lower odds of this phenotype, whereas no clinical variable was independently associated with increased odds. These findings highlight the importance of interpreting non-obstructive coronary anatomy after fibrinolysis as a post-treatment angiographic phenotype rather than assuming a single underlying mechanism.

Declarations

Author Contribution

V.A.C.N.: Conceptualization, methodology, investigation, data curation, formal analysis, interpretation of data, project administration, visualization, writing – original draft, and writing – review and editing.J.M.A.S.: Formal analysis, statistical analysis, interpretation of data, and writing – review and editing.M.T.S. and S.R.B.F.: Investigation and data collection.P.I.D.M.M.: Investigation and data collection.Â.A.V.P.: Writing – review and editing.A.H.P.B.: Conceptualization, supervision, project coordination, and writing – review and editing.All authors read and approved the final manuscript.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request, subject to applicable institutional and ethical restrictions.

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Figures

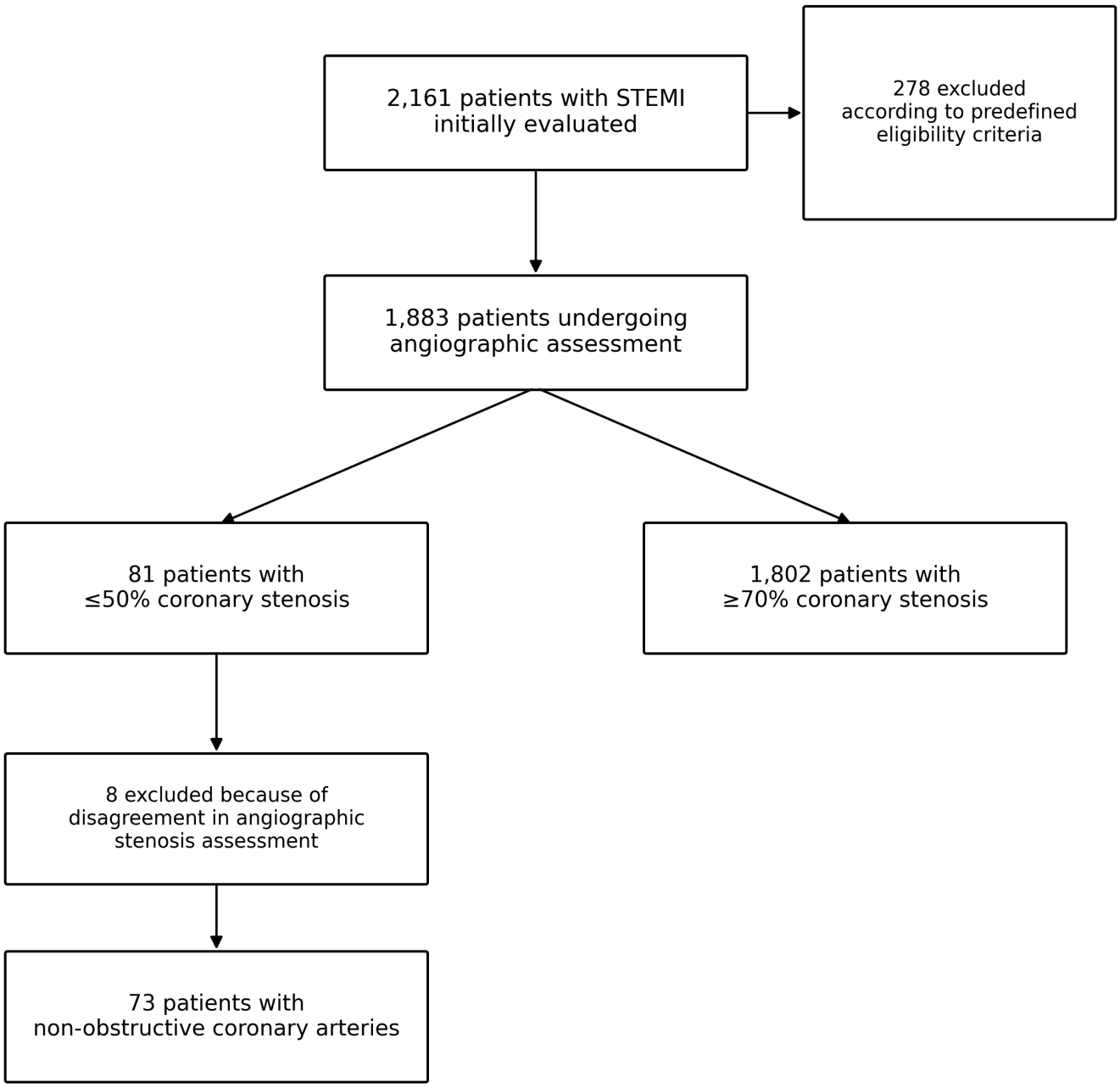


Figure 1

Patient selection flowchart

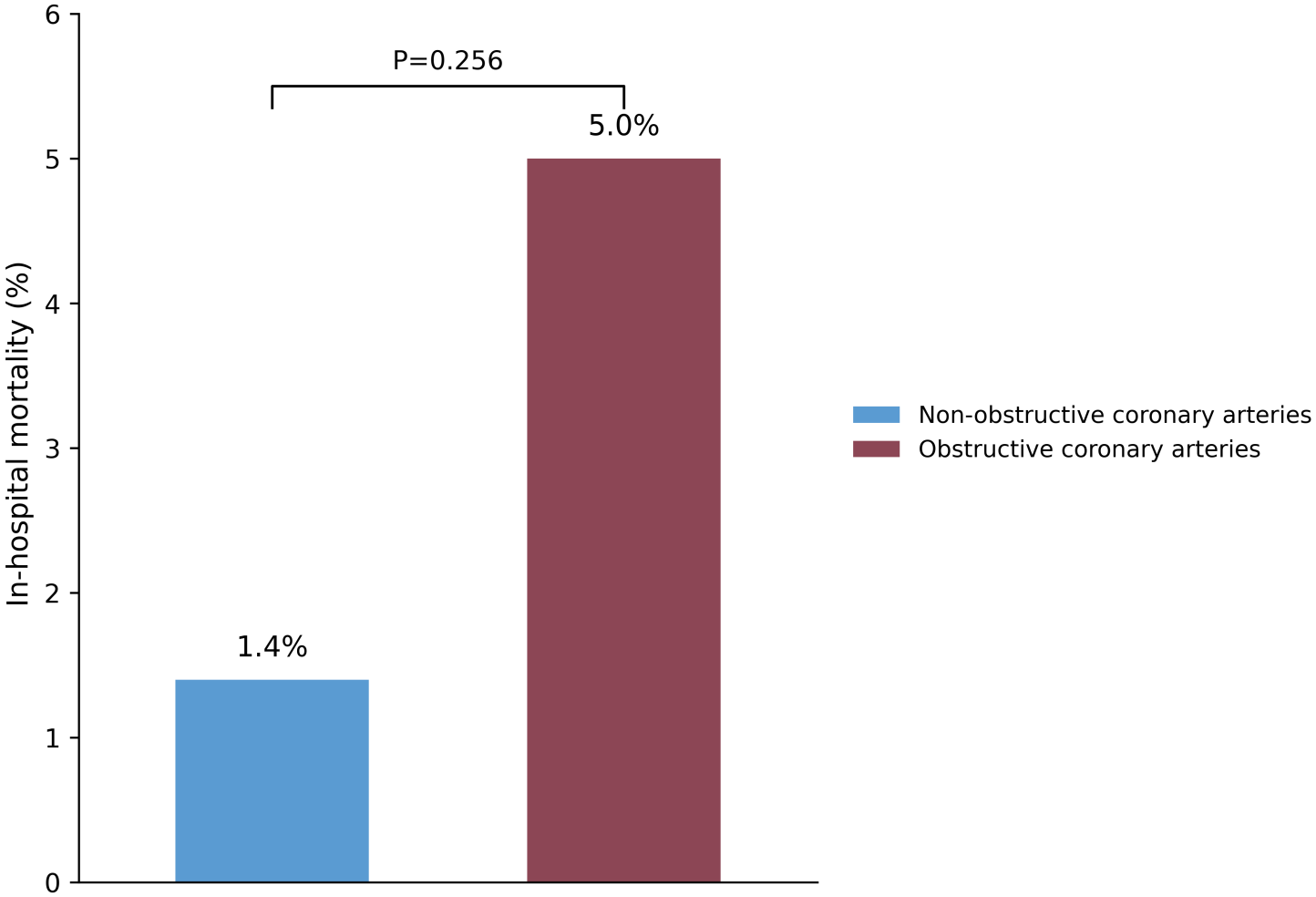


Figure 2

In-hospital mortality according to coronary anatomy after fibrinolysis.

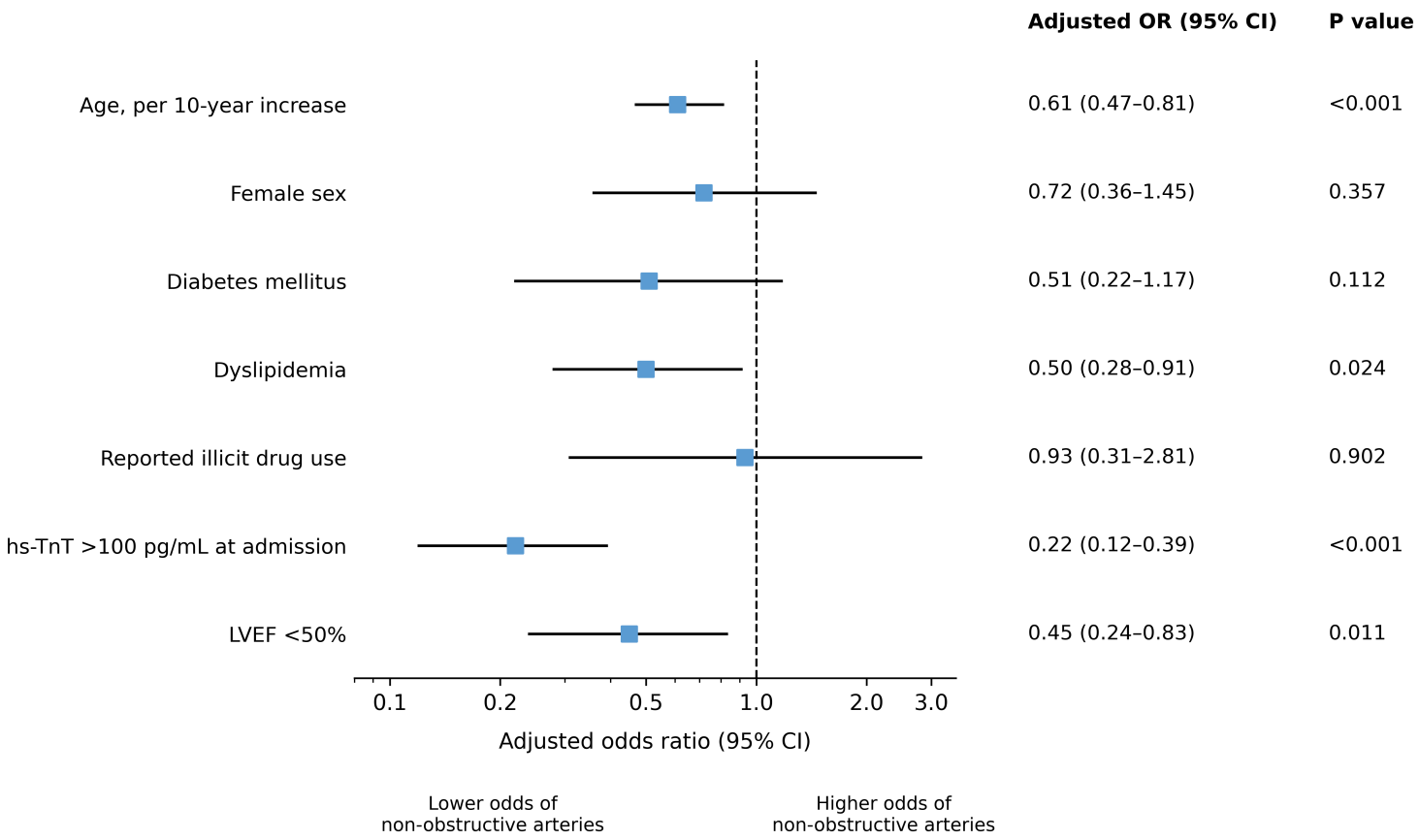


Figure 3

Multivariable analysis of factors associated with non-obstructive coronary arteries after fibrinolysis

Supplementary Files

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- Graphicabstract.png